Journal

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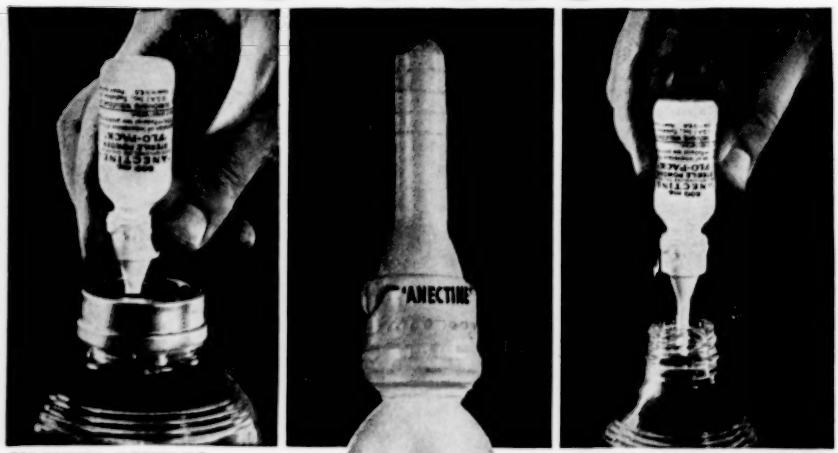

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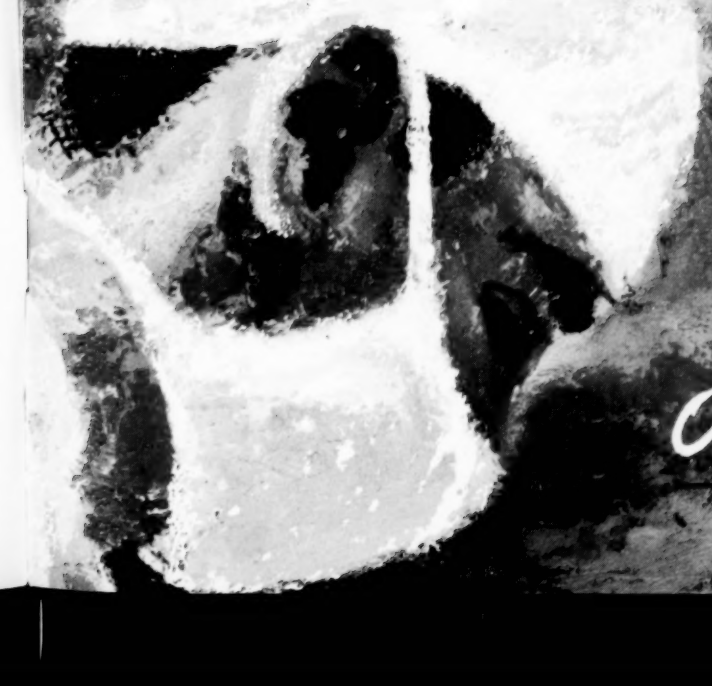

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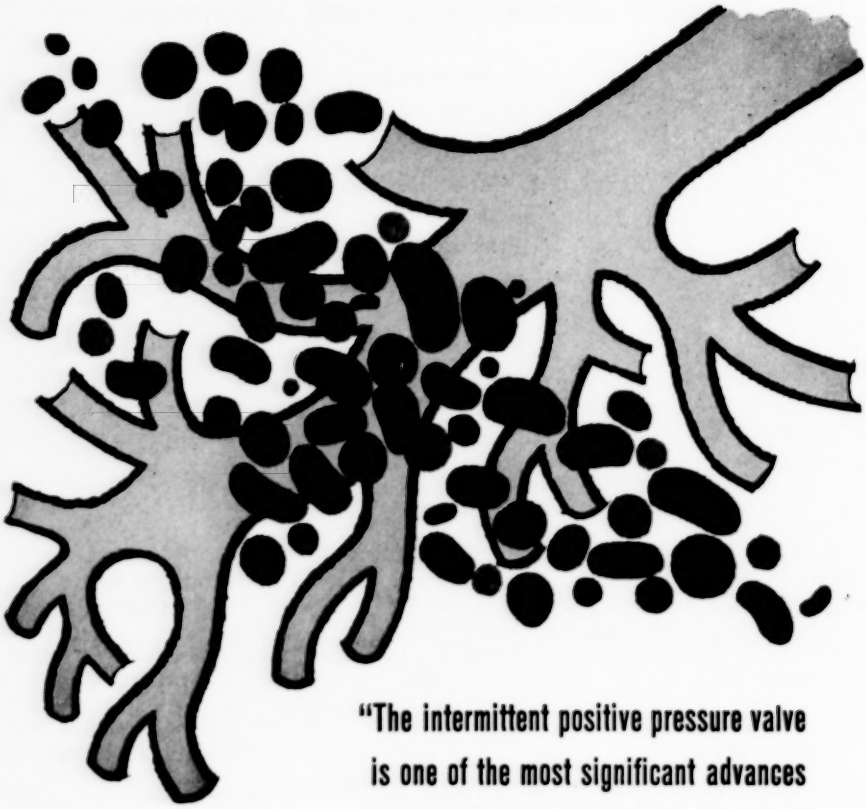

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THE PARENTERALS PAGE NUMBER 7

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In general, the normal infant's heat and urine output is proportionately double that of an adult. His body surface for perspiration loss is also doubled. Hence disease-induced water losses are particularly dangerous. In severe diarrhea, for example, the infant may quickly lose up to a quarter of his body weight.

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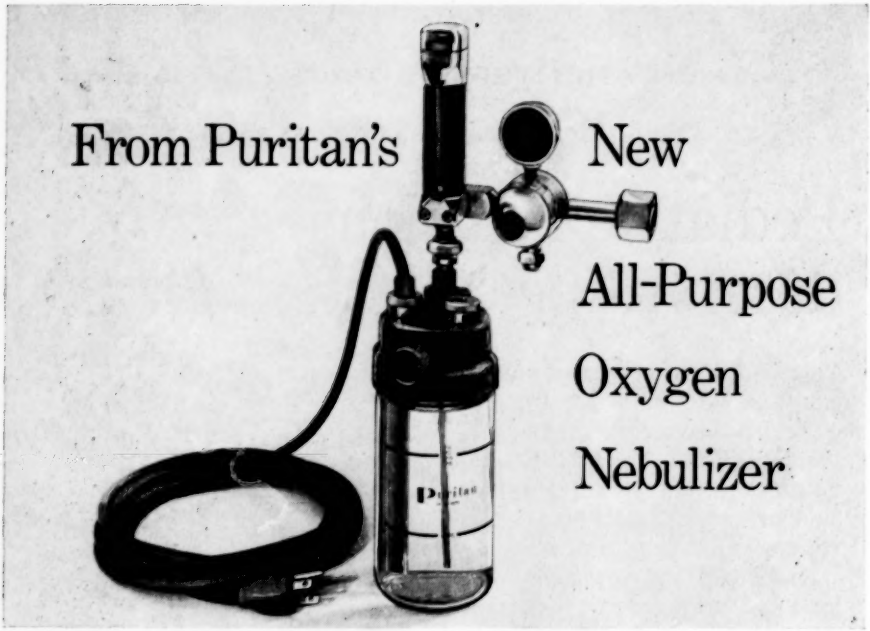

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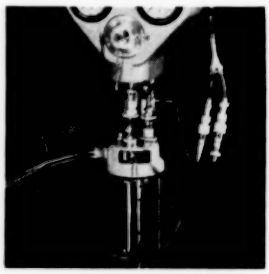

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Vagal Reflexes

Helen P. Vos, C.R.N.A., B.S.*
St. Louis, Missouri

Many body reflexes are not clearly understood and have been given various theoretical explanations. Observations and the response to drugs have led to some of our present day theories, whereas the pathways of some reflexes have been proved by extensive research in the laboratory. I have tried to compose a brief review of the work of many authors to place the vagus nerve in its proper perspective.

The *central nervous system* is composed of brain and spinal cord. Nerves going toward or from the brain are called the cranial nerves of which there are 12 pairs. Those going toward or from the spinal cord comprise the 31 pairs of spinal nerves. The cranial and spinal nerves are classified as the *peripheral nervous system.*

FUNCTION OF THE NERVOUS SYSTEM

The function of the entire nervous system is adjustment to the external and internal environment. This may be summated briefly as follows:

I. Function: environmental adjustment
  A. External environment: Voluntary Nervous System
    1. Afferent (sensory) — gives us sensory awareness
    2. Efferent (motor) — conscious movement of somatic muscle
  B. Internal environment: Involuntary or autonomic nervous system
    1. Afferent (visceral)—usually without sensation
    2. Efferent (visceral)—alterations in movement of smooth muscle or myocardium. In some instances promotes or inhibits secretions.

From the outline below one can infer that some functions of the nervous system are at the level of consciousness or awareness, but many are below the level of consciousness.

Those reflexes dealing with external environment are anesthetized with ease and rapidity according to the particular drug used. Most signs of the level of anesthesia deal with the degree of anesthesia to the voluntary nervous system; one should be cognizant of the fact that general anesthesia applies to both the sensory afferent fibers and the motor efferent fibers. The muscle relaxants are not anesthetic drugs. They have no effect on the sensory afferent nerves. Their effect is produced at the junction of the efferent motor nerve and the muscle it innervates.

Desirable general anesthetic drugs will adequately anesthetize the voluntary nervous system but will leave the involuntary or autonomic nervous system to perform its function, namely regulating the internal environment.[2]

The combination of nerve fibers forming the cranial and spinal nerves are varied. Some are only afferent sensory, such as the optic or 2nd

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Presented at the Institute for Nurse Anesthetists, St. Louis, January 16, 1961.

cranial nerve, whereas some are a combination of two, three or four of the nerve fibers listed above. The vagus nerve or 10th cranial nerve is one containing all four types of fibers.[1]

AUTONOMIC OR VEGETATIVE NERVOUS SYSTEM

Before we become involved in discussion of the reflexes of the vagus nerve, a brief look at the function and distribution of the autonomic nervous system will make the vagal reflexes more readily recalled. There are two divisions: the sympathetic or thoracolumbar and the parasympathetic or craniosacral. One generally thinks of the fibers as being only efferent but this is not totally correct.[3] However, we will speak primarily of the efferent fibers. In life, as well as during the trauma of surgery, the sympathetic nervous system helps during stress. When the stress situation is removed the parasympathetic system restores the internal environment to normal conditions.

What signs do we observe in sympathetic and parasympathetic stimulation? Using the table in Fig. 1, think of the sympathetic system as a tiger lying under a tree eating his catch. Suddenly he picks up the scent of an approaching lion and he knows he is in immediate danger. Instantaneously the following occur:

Pupils—dilate to improve vision.
Salivary glands—flow is inhibited.
Blood vessels in striated muscle—dilate to give good blood flow.
Bronchi—dilate to permit more oxygen intake.
Heart rate—increases to improve circulation.

SYMPATHETIC		PARASYMPATHETIC
Dilate	**Pupils**	Constrict
Flow inhibited	**Salivary glands**	Flow increased
Dilate	**Blood vessels (striated muscle)**	Possible slight constriction
Dilate	**Bronchi**	Constrict
Increases	**Heart Rate**	Decreases
Dilate	**Coronary vessels**	Constrict
Secrete adrenalin	**Adrenal glands**	No innervation
Glycogenolysis	**Liver**	Secretory
Inhibited	**Digestion**	Peak performance
Constricted	**Blood vessels in thorax & abd.**	Dilated
Vasoconstriction	**Kidney**	Vasodilatation (slight)
Constricts	**Spleen**	Stores RBC

Fig. 1. Physiology of sympathetic and parasympathetic nervous systems.

Coronary vessels—dilate to supply more nutrition and oxygen to the myocardium.
Adrenal glands—secrete more adrenalin which in turn keeps stimulating the sympathetic nervous system.
Liver—changes stored glycogen to glucose and ejects it into the blood for quick energy.
Digestion—is inhibited or may even discontinue.
Blood vessels—in the thoracic and abdominal viscera are constricted. Blood is not needed by the digestive system at this time.
Kidney—vasoconstriction so most of the blood will by-pass the kidney.
Spleen—constricts, forcing its stored red blood cells into circulation.

The animal is ready "for fight or flight"!

On the other hand, we have the actions of parasympathetic system. Try to picture a contented cow chewing her cud under a tree. We would observe the following:

Pupils—constrict. She has nothing to look or search for.
Salivary glands—secrete in abundance. This is necessary for the beginning of digestion.
Blood vessels—in striated muscle will be somewhat constricted due to lack of exercise.
Bronchi—partially constricted. No need for increased respiratory exchange.
Heart rate—normal or somewhat decreased.
Coronaries—normal or constricted for the heart is not being taxed.
Adrenal glands—probably do not have innervation by the parasympathetic nerves.
Liver—collects the excess glucose from the blood (result of digestion) and stores it as glycogen. It also secretes many enzymes necessary for digestion.
Digestion—at its peak performance.
Blood vessels—in the splanchnic bed are engorged with blood. This is necessary for two reasons; supplying the digestive tract with blood and picking up nutrient material from the small intestine.
Kidney—normal blood flow or some vasodilatation.
Spleen—dilates and again stores red blood cells. This system permits restoration and conservation of body energy.

Neurohumoral System: Certain chemicals are necessary for nerve impulses to become effective. Very briefly, one may say acetylcholine is the humoral transmitter for parasympathetic nerves and is readily destroyed or hydrolized by the enzyme acetylcholinesterase to prevent sustained effects.[3] The humoral transmitter for sympathetic nerves is noradrenalin which is quickly oxidized by amine oxidase.

Innervation: Most viscera is innervated by both sympathetic and parasympathetic nerves. One may consider them as being antagonistic to each other. The effects on the physiology will depend upon the degree of stimulation or depression of one system. This is illustrated in Fig. 2. The midline indicates the normal balance. Four changes may alter the normal but produce only two responses.

One of two things may give the appearance of sympathetic stimulation. Either the parasympathetic system may be depressed, as by atropine, or the sympathetic system may be stimulated, as in stress. On the other hand, two separate drugs or environmental changes may give the symptoms of an overactive parasympathetic system, e.g., a depressed sympathetic system as may be seen during high spinal anesthesia, or stimulated parasympathetic system produced by reflexes or the administration of physostigmine.

VAGUS NERVE AND REFLEXES

The vagus nerve is the 10th Cranial Nerve. The cell bodies or nuclei of its efferent fibers lie in the medulla oblongata. The vagus is classified as a mixed nerve because it has fibers belonging to all four classifications. Some are afferent sensory, some efferent motor, others afferent visceral and still others, efferent visceral. The

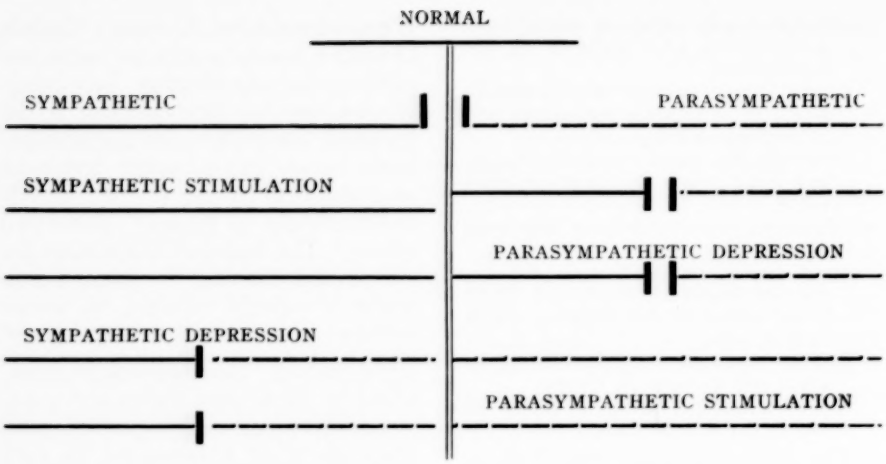

Fig. 2. Effect of stimulation or depression of either of the autonomic nervous systems.

word "Vagus" is Latin for "wandering", and the nerve is well named. This is demonstrated by the diagram in Fig. 3, where briefly we show where the receptors are found for the afferent fibers and where the effectors are found for the efferent fibers.

Vagal reflex is an impressive term. When we need an explanation for some otherwise unexplained alteration in physiology, vagal reflex often calms the most arrogant and argumentative individual. Any surgical procedure could cause a vagal reflex. Usually,

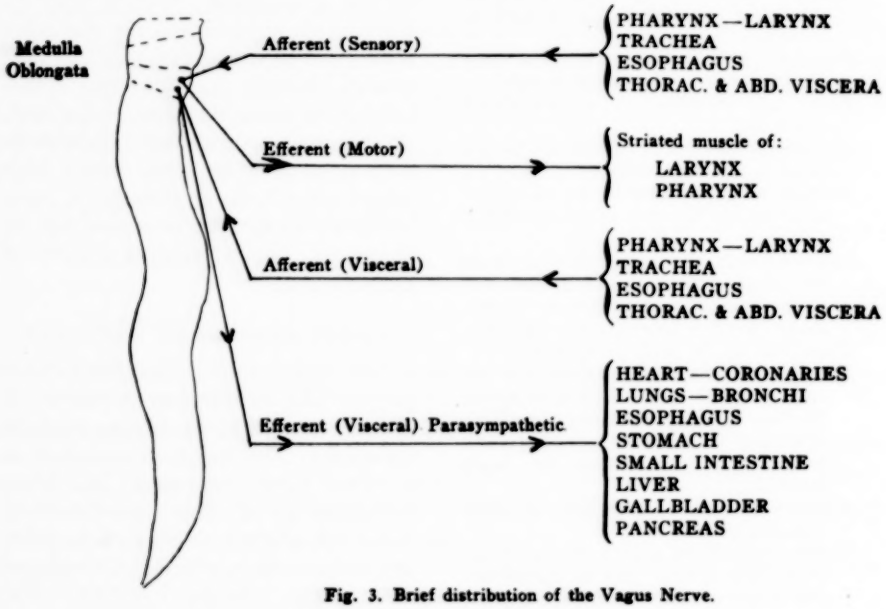

Fig. 3. Brief distribution of the Vagus Nerve.

when the term is used, we mean that the fiber which is classified as part of the parasympathetic system has been overly stimulated, resulting in bradycardia, hypotension or bronchial spasm. Actually vagal reflexes are numerous and varied. We shall mention only a few of the more common of them encountered during anesthesia.

When we wish to expose the glottis of a conscious patient, we follow the procedure necessary for adequate topical anesthesia of the oral and laryngeal pharynx. Again referring to Fig. 3, we see that if topical anesthesia is inadequate, the receptors of the afferent sensory fibers will receive the stimulus and convey it to the medulla. The efferent motor fiber will probably cause the striated muscle of the larynx and pharynx to respond. Gagging and spasm result. Coughing occurs as well, but this is not accomplished through the vagus nerve. The only part the vagus plays in the cough reflex is the closure of the vocal cords, permitting the muscles of the chest and diaphragm to build up pressure behind them. Closure of the glottis is caused by striated muscle—we can, to an extent, control it. Therefore, good topical anesthesia will not permit this reflex to have a starting point and this is to be desired.

To illustrate another reflex—An endotracheal tube has been inserted with rather inadequate general and topical anesthesia. The trachea is still sensitive. The anesthetist inflates the cuff and bradycardia results. Impulses may traverse either the afferent sensory or afferent visceral fibers (or both), be transmitted to the neuron forming the efferent visceral fiber. This may produce bradycardia and probably hypotension as well. This reflex may be called vago-vagal. This supposedly signifies that both the afferent and efferent neurons causing the reflex are vagal fibers. Do note the word "may". A rather recent study shows that tachycardia and hypertension usually occur.[4] However, bradycardia and hypotension have also been observed. It is also noteworthy to remember that this reflex (parasympathetic stimulation) may cause constriction of the coronary vessels and insult the myocardium further by producing anoxia. The disastrous consequence of an anoxic myocardium might well be ventricular fibrillation.

Bradycardia and hypotension occasionally occur in patients undergoing some type of ocular surgery. The trigeminal (5th cranial) nerve furnishes the afferent fiber and the visceral vagus fiber produces the effect.[2] Pressure of the face mask on the eye, or stimulation of the nasal mucosa under light anesthesia may also produce the same effect. Usually this is not extreme, but could be avoided by topical anesthesia to the eye, effective nerve block, reducing the pressure or stopping the stimulation.

Whenever intentional or inadvertent pressure is applied to carotid sinus in the neck, parasympathetic stimulation usually follows. The degree of hypotension and bradycardia will depend a great deal on the sensitivity of the carotid sinus.

The efferent fibers of the vagus are very numerous in the viscera of the thorax. During intrathoracic surgery these fibers may be stimulated directly, therefore, not requiring an afferent pathway. Stimulation of the vagus just above or at the level of the heart may cause cardiac standstill and absence of blood pressure.[2]

We know that pain can produce shock. Often this picture of shock differs from shock produced by blood loss by bradycardia instead of tachycardia accompanying the hypotension. The skin may be warm and dry; not cold and clammy. One good example of this may be the patient in the recovery room who has had a hemorrhoidectomy under spinal anesthesia. After the effects of the spinal anesthetic drug have worn off, the pain may be intense. The pelvic nerve forms the afferent pathway which gives the patient a sensation of pain but it may stimulate the parasympathetic nerves of the vagus as well.[2] Parasympathetic stimulation may be observed during various pelvic and rectal procedures, particularly if the patient is maintained in a light plane of anesthesia, with or without muscle relaxants. I have witnessed a cardiac arrest during a proctoscopic examination in which no anesthetic had been given and, unfortunately, no premedication had been given either. I am not prepared to say that this was due to vagal reflex, but no better explanation was volunteered. I have also observed sudden and intense bradycardia and hypotension during hysterectomy. Apparently, again, the pelvic nerve produces the afferent pathway and the vagus parasympathetic fiber, the efferent pathway. In almost every instance an intravenous injection of atropine grs. 1/150 gave dramatic improvement.

POSSIBLE PREVENTION AND SUGGESTED TREATMENT

How may one avoid these bothersome reflexes? Adequate oxygenation with proper elimination of carbon dioxide is of paramount importance. Deficient ventilation with a high concentration of oxygen may meet the metabolic needs for oxygen but may not permit elimination of carbon dioxide. This tends to have an acidifying effect, lowering the blood pH. It has been fairly well established that this prolongs the action of parasympathetic stimulation by inhibiting the hydrolysis of acetylcholine by acetylcholinesterase.[5]

Since the advent of muscle relaxants we usually maintain patients in the 1st plane of the surgical stage. This is generally desirable. However, this level of anesthesia is often inadequate to completely block all afferent fibers, neither the afferent sensory or the afferent visceral, and it is possible for some stimuli to initiate the reflex. The efferent visceral pathway is not affected by anesthetic drugs until the 3rd and the 4th planes of the surgical stage are reached. This level of anesthesia the patient cannot tolerate, at least not for any length of time. One very effective method of correction is the use of regional drugs at the receptor cells of the afferent fiber. The reflex does not "get a start" so to speak. Topical, infiltration or nerve blocks are very effective. One might ask for less stimulation to a part, e.g., removal of instruments pressing the carotid sinus, but not all stimulation can be avoided.

Atropine blocks or inhibits muscarinic effects. That is, the effector cells in the viscera which are innervated by the parasympathetic nerves are inhibited. The entire reflex arc is permitted to be formed until it reaches the cells in the viscera where no effect is permitted. The end result gives the appearance of no reflex having ever been started.

One point about atropine needs clarification. For many years the use of atropine before Pentothal Sodium anesthesia was stressed. It was

thought to prevent laryngospasm. Atropine does have a drying effect on the salivary glands, thereby decreasing the possibility of mucous and saliva stimulating the vocal cords, but, atropine probably has no effect on striated muscle. Laryngeal spasm is made possible by the striated muscles of the larynx. The muscle relaxants, which paralyze striated muscle, will prevent or correct laryngospasm, but with our present knowledge of the nervous system and atropine, the usefulness of atropine for this purpose seems doubtful.

One cannot expect the muscle relaxants to prevent vagal reflexes. The curariform drugs may block the vagal reflexes at the autonomic ganglia but dosage must be equivalent to that required to produce respiratory muscle paralysis. However, the synthetic curariform drug, Gallamine (Flaxedil) seems to cause little or no autonomic ganglionic blockade.[2]

SUMMARY

A brief review of the physiology of the autonomic nervous system has been given emphasizing that the sympathetic system prepares the body for "fight and flight" and the parasympathetic in turn restores and conserves energy.

Various vagal and vago-vagal reflexes have been discussed and the afferent and efferent pathways have been outlined. These reflexes may or may not occur. In the event that they are present they may be recognized by bradycardia (including possible coronary constriction), hypotension and at times bronchiolar constriction.

Suggested prevention and treatment is the use of topical, local infiltration, and nerve block anesthesia; deeper planes of anesthesia, atropine and possibly large doses of the curariform drugs with the exception of Flaxedil.

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New Drugs and Techniques in Anesthesia

Gretchen Guernsey, M.D.*
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Superficially, anesthesia has not changed much in the last twenty years, and yet new drugs, new techniques, or new approaches to old drugs and techniques are constantly being introduced. The result of all this activity is that anesthesia is gradually but steadily changing. Any new drug comes with the claims of its manufacturer which are naturally optimistic, as the drug represents a considerable investment of time and money before it is introduced. This is followed by articles in the literature which are usually favorable because they are written by people who are, by nature, progressive and enthusiastic. Then comes a rash of articles, reporting side actions or failure to duplicate the results of the earlier papers and, eventually, the drug settles into its rightful place or drops from sight.

NEW INTRAVENOUS ANESTHETICS

One of the new anesthetic agents which seems to offer the most promise is methohexital, presented by Lilly under the trade name Brevital. Compared with other intravenous barbiturates, it is two to five times as potent, has a duration about half as long, and affords more prompt recovery, even after large doses. It has less cumulative action than thiobarbiturates. It is not deposited in fat as the thiobarbiturates are, so maintenance requires large dosage in proportion to potency. It is more rapidly metabolized than the thiobarbiturates. It is incompatible with acid solutions such as atropine sulfate, dimethyl tubocurarine iodide and succinylcholine. Hiccoughing and mild laryngospasm occur occasionally. They are relieved by additional amounts of methohexital. Apnea is rather frequent, it is relieved by oxygen under intermittent positive pressure. There is an occasional fall of systolic pressure. With large doses (7.5 times the mean anesthetic dose), apnea, hypotension, and tachycardia were observed and yet smooth awaking occurred in about twenty minutes. Methohexital may be used with any of the recognized premedicating agents, inhalation anesthetics and muscle relaxants. The 1% solution is irritating and if it is extravasated, moist heat may be necessary.

There have been several non-barbiturate intravenous anesthetics, Viadril, Dolitrone and SCTZ, none of which seem to have gained any wide acceptance. Sernyl is another one which has pretty much disappeared from view, and this was due to its tendency to produce psychic disturbances. It is a cyclohexylamine derivative which is capable of producing sensory blockade of sufficient depth to permit completion of minor surgical procedures

* Chief, Anesthesia Department, Providence Hospital, Kansas City.
Presented at the Institute, Kansas Association of Nurse Anesthetists, Kansas City, November 12, 1959.

without concomitant sleep and without depression of respiration and circulation. Cyclohexamine, a related compound, is now being studied. It permits endotracheal intubation, but the patient bucks and has to have either nitrous oxide or a short-acting barbiturate. It permits minor surgery, but does not cause muscular relaxation. It is accompanied by psychic phenomena which are at times very unpleasant and the patient should have nitrous oxide or a little thiopental.

Intravenous Doriden produces adequate sleep of short duration with minimal respiratory depression but good obtundation of pharyngeal reflexes. The solubility problem remains to be solved.

Anileridine is being used in a concentration of 0.3 mg. per cc. intravenously by continuous drip, supplemented with thiopental, nitrous oxide and curare. The average patient will take 30 to 60 mg. per hour. This produces smooth anesthesia and rapid awakening but requires minute-to-minute alertness on the part of the anesthetist.

There are still occasional articles appearing in the literature recommending intravenous procaine as an anesthetic agent. These come mostly from South America where nitrous oxide is difficult or impossible to obtain and they must look elsewhere for analgesia. Now intravenous lidocaine is being suggested. It is given in 0.1% solution and this may be combined with 0.5% succinylcholine. Use of lidocaine reduces the requirement of thiopental by 13%, and, especially, for major surgery, it definitely smooths out the anesthesia. No claim is made that the need for postoperative morphine is decreased, or that the incidence of nausea and vomiting is any less.

MUSCLE RELAXANTS

Muscle relaxants are classified as depolarizing (decamethonium, succinylcholine, suxethonium [Brevidil E]), and non-depolarizing (d-tubocurarine, Flaxedil, Laudolissin, Myotolon) according to their action on the motor-end plate. One of the new ones is Prestonal, a short-acting apparently non-depolarizing relaxant. It was very successful, used in a single dose for intubation or to supplement the action of curare late in the operation. Intravenous drip was associated with the greatest incidence of side effects, which are elevation of blood pressure and pulse rate and prolonged apnea. Tensilon and neostigmine do not antagonize it.

Hexafluorenium is a bis-quaternary ammonium compound which is non-depolarizing. It is recommended to be given in a dose of 0.5 mg. per Kg. then followed by a drip of 0.05% succinylcholine. Intubation is done when relaxation is adequate. The drip of succinylcholine is continued. When relaxation lightens, additional doses of Hexafluorenium 0.2 to 0.3 mg. per Kg. are administered. This combination reduces the required dose of succinylcholine to one tenth or less of the expected dose. Prolonged apnea is not seen and the muscle twitching seen with succinylcholine alone does not occur.

Imbretil is a synthetic long-acting relaxant which is very popular in Europe and is beginning to be used in this country. It produces neuromuscular blockade by depolarization initially; after an hour or so, the block becomes like curare block and is antagonized by neostigmine. Some skeptics say this is not so, that the reason neostigmine appears to reverse the block is that the Imbretil is wearing off. Thiamine reverses the block when given less than forty minutes

after the Imbretil. Intravenous Imbretil produces apnea in 45 to 180 seconds, abdominal relaxation in three minutes. Usually, a dose of 0.6 to 0.8 mg. per Kg. will provide adequate relaxation for one and one-half to three hours of surgery. The relaxation is the most profound imaginable, with the intestines contracted. If additional relaxation is required toward the end of the operation, succinylcholine should be used. Also, intubation should be done under succinylcholine to keep down the total dose of Imbretil, as larger doses may cause prolonged apnea. Imbretil appears to be synergistic with succinylcholine and with ether. It liberates histamine, but less so than curare. Bronchospasm has not been reported. Hiccough or diaphragmatic "flapping" occurs in about 8%, but is easily treated with succinylcholine. Burroughs-Wellcome drug 49-204 antagonizes decamethonium but potentiates the action of Imbretil.

A recent article reports the occurrence of severe bronchospasm in six successive cases given a combination of Mylaxen and succinylcholine. Mylaxen is not being used very widely, but it was first reported to have the advantage that it would not produce relaxation in conscious patients or that the relaxation would disappear as the anesthetized patient regained consciousness. Then it was found that relaxation was not adequate for most abdominal surgery, and it was recommended that Mylaxen be combined with succinylcholine. The first report was that the combination caused a minimum of side effects, but apparently it may be quite dangerous.

PREMEDICATING AGENTS

One of the most fertile fields of drug research has been in premedicating agents. Most people are now using one of the ataractic drugs whose name is legion. To run through them briefly by classes: The Rauwolfia alkaloids have been unsuccessful as premedicants, as they are primarily hypotensives and do not produce sedation with a single dose. The phenothiazine derivatives include Phenergan, Pacatal, Vesprin, Sparine, Compazine and Dartal. Of these, Phenergan is being widely used and Pactal is coming into use. They appear to reduce the requirement of preoperative narcotic, reduce the severity of undesirable reflexes (laryngospasm and bronchospasm), reduce the barbiturate or ether requirement during surgery, reduce the incidence of postoperative vomiting and reduce the postoperative narcotic requirement.

The Benzohydrol compounds include Meratran, Suavital and Atarax. These are not at present being used for premedication except perhaps in isolated instances, although Atarax has been recommended for this purpose by its producer. The Carbamates are represented by Miltown, and Valmid. Miltown is more useful postoperatively in a patient who has become disturbed than preoperatively. Valmid is a nonbarbiturate sedative to be used as you would use an oral barbiturate. Other nonbarbiturate sedatives are the acetylinic compounds Dormison and Placidyl, and the piperdine derivatives Nolundar and Doriden.

Benadryl, although it is by no means new, deserves mention in this classification because it is a tried-and-true mild sedative.

There are several new analgesics on the market. Anileridine has had rather extensive use and is recommended by the fact that it is as potent orally as intramuscularly, as compared to meperidine, which is

only half as potent when given by mouth. As for narcotic potency, it is about twice as potent as meperidine. Levodromoran, although it is not new, should be mentioned. It has very little psychic effect, but is useful in patients who are sensitive to other narcotics or in whom respiratory depression is to be avoided. Dipipanone which is called Pipadone, seems to lack the hypnotic effect of Demerol and has not been successful as premedication or as an analgesic in labor.

Phenampromide (Lederle) is a potent analgesic agent, but has not been felt to have enough advantage over present agents to merit commercial production. Phenazocine (SKF) is thought to deserve trial as a premedicating agent although it tends to depress respiration.

Winthrop has a series of non-narcotic analgesics which are about thirty times as potent as meperidine but only five times as toxic. So far, they are known only by number. Win 13 produces more euphoria, Win 14 and 16 are better for postoperative pain. Win 13 and 14 if used for premedication, may cause excitement on awakening, but this is relieved by another dose of the same drug. Endo Laboratories have a new morphine derivative called Numorphan, which they claim provides analgesia superior to that of morphine and in smaller dosage, has a more rapid onset of action, and has a notable lack of the side effects of morphine.

There is a host of anticholinergic drugs, none of which will ever take the place of scopolamine for premedication. There are Banthine, Prantal, Antrenyl and Pamine. Most or all of these were developed for treatment of gastric or intestinal spasm. For this reason, they are designed to have as little sedative and as little drying effect as possible, which minimizes their usefulness as premedicating agents. A new one is phenglutarimid or Aturbane, which is a potent depressor of secretions, but lacks the sedative properties of scopolamine. A recent Danish paper concluded, after a comparison of various premedicating combinations, that morphine and scopolamine provided best sedation, best amnesia, and best control of secretions.

A fairly new anti-emetic agent is Trilafon (perphenazine). It is 16.6 times as effective as chlorpromazine against the emesis induced by apomorphine. As the emetic stimulus is increased, the relative efficacy of Trilafon seems to increase. Roche has a new anti-emetic called Tigan which is not now available in parenteral form, so its usefulness would be limited to perhaps giving it preoperatively to a patient who gives a history of nausea after previous anesthetics.

Not much is being heard from the field of narcotic antagonists. We still have Nalline and Lorfan to antagonize the respiratory depression due to opiates and synthetic narcotics. The most recent work has shown that they also decrease analgesic effects, although not to the same extent as respiration.

The drugs now being used to combat barbiturate depression are Megimide and Ritalin. They both stimulate respiration and reflex irritability as well as return toward consciousness, but Ritalin may raise the blood pressure and cause transient retching especially if the patient is not very deeply asleep. A small dose of Ritalin (0.05 mg. per pound) added to the usual dose of Megimide has the effect of doubling the dose of Megimide and also inhibits the tremors commonly seen with Megimide alone.

TECHNIQUES

There is an interesting recent report on blood transfusions. A total of 436 transfusions was given to 105 patients, each of whom had had previous blood transfusion problems. Immediately before transfusing, 50 or 100 mg. of soluble prednisolone was introduced directly into the blood. In every case the steroid effectively prevented minor hemolytic and/or pyrogenic reactions. Also, when the steroid was injected intravenously after reaction had occurred during transfusion, it afforded effective treatment. There is some thought that transfusion reactions may be due to the agglutination activity of the donor leukocytes.

There have been two articles in the recent literature regarding intubation in the lateral position for patients who are to be operated on in that position. It has the advantages that it avoids hypotension due to change of position, decreases anesthesia time, provides opportunity to obtain baseline assessment of the patient's tolerance of the lateral position, minimizes need for assistance from the operating room personnel in positioning the patient and minimizes displacement of intravenous needles.

Some use is being made of cuirass respirator with thiopental and succinylcholine for laryngoscopy and bronchoscopy. This is theoretically ideal if one can be sure the ventilator is doing its job. It must fit the patient well and ventilation may not be adequate in emphysematous or very obese patients. A more satisfactory solution in many cases is to do the laryngoscopy or bronchoscopy with an endotracheal tube in place.

Endotracheal tubes are about to be standardized by a committee representing all the agencies having an interest in such standardization. Their recommendations follow:

1. Endotracheal tube shall be the name.

2. The two ends shall be called the tracheal end and the machine end. The cuff shall be called an endotracheal cuff and the tube by which it is inflated shall be called an inflating tube.

3. The metric system shall be the standard of measurement. The size of the tube shall be the internal diameter in millimeters. Consecutive sizes shall increase by 0.5 mm. Oral tubes shall be produced with a radius of curvature of 14 cm. and a bevel angle of not less than 30°. The bevel may face either right or left.

4. Endotracheal cuffs may be either slip-on or permanently bonded. The mounted cuff shall expand symmetrically to at least one and one-half times the external diameter of the endotracheal tube. Sizes shall indicate the internal diameter of the unmounted cuff expressed in millimeters. The length of the inflatable portion shall be in proportion to the size, according to a table of standards. The external diameter of the inflating tube shall not exceed 0.3 mm. It shall come off the cuff at a flat angle without undue projection.

5. Marking materials shall be of contrasting color, non-toxic, compatible with human tissue and resistant to cleansing and sterilizing agents. The tube shall be marked with the word oral or nasal and with the size. The cuff shall be marked with the name, the size, and the range of endotracheal tubes it will fit.

6. Materials should be nontoxic, compatible with human tissue, resistant to chemical cleansing and sterilizing agents, and to autoclaving and anesthetic agents. The surface of the tube should be smooth externally and

internally and the surface of the cuff externally. The tube should be rigid enough to hold its curve, flexible enough to conform to the patient's anatomy.

There is developing some use of ethylene oxide for sterilizing anesthetic and operating room supplies. It is explosive in a range of 3% to 100% in air, is rendered non-explosive by adding CO_2. It is bactericidal, not bacteriostatic. It passes through polyethylene, rubber, etc. It is expensive so you run it only once a day which means you must have enough supplies on hand for one day.

CONCLUSIONS

A few of the new ideas in anesthesia are presented. While it is recognized that not all of them are new to everybody, it is hoped that the presentation may help to counteract the feeling that we all have, occasionally, that our specialty is in a hopeless rut.

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The Role of the Adrenal Medulla During Anesthesia and Surgery

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A paradox exists in the realization that most complications occurring during surgery and anesthesia and in the immediate 72-hour postoperative period which the anesthetist is concerned with are not the direct result of the administration of anesthetics to the patient. For the most part, they are the result of alterations in physiological dynamics. The improper selection of pharmacologic compounds (anesthetics), however, can additionally influence these physiologic dynamics. It is the consuming duty of the anesthetist to be ever aware of the implication of all possible factors in the surgical-anesthetic experience that may involve influencing elements in the production of complications.

From a review of the distribution of complications occurring in the immediate 72-hour postoperative period at St. Vincent's Hospital for the year 1959, it was evident that the outstanding difficulty which one must of necessity deal with is hypotension —a subject which has been thoroughly explored and discussed in all schools. For the purpose of this discussion we are including in this category all cases that exhibited any drop in blood pressure on the one hand and at the extreme end of the range, all those that would ordinarily be included as "shock".

Hypotension has all too frequently been thought of in immediate post-anesthesia as a direct result of the withdrawal of some anesthetic from the patient such as the hypotension that follows the administration of cyclopropane. This cycle is present in our series of cases of hypotension but it is not the outstanding cause. I should submit that the outstanding cause is oversight at some point in the anesthetic management of the patient, and that this oversight is all too often *not* the result of personal negligence but has been the result of obscure or hidden physical signs that the patient may exhibit at certain important times. If they were more demonstrable, they could easily be submitted to proper medical management and would no longer be implicated as one of the major contributory causes of further patient morbidity and mortality.

In assessing the role of hypotension in this regard, one must perpetually consider the patient as a delicate scale which is brought into balance by the correct interplay of the fluids, of the cardiac function, of the tone and response of the peripheral vascular system, of the response of the

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nervous system and numerous chemical substances which may affect any of these according to their qualitative and quantitative admission to the circulation by glandular structures or medical administrations. We are all aware that the premedicated or anesthetized individual will develop hypotension readily by such things as changes in posture, by oligemia, by inadequate cardiac action, and by disturbances in the central and autonomic nervous systems. But we all too frequently forget the important stabilizing mechanisms brought into play, including the outpouring of hormonal substances—in particular, those associated with the adrenal gland.

The contribution of the adrenal in maintaining normal blood pressure is biphasic. On the one hand those substances, the steroid compounds, that are secreted by the adrenal cortex are essential in the distribution and maintenance of body fluids and electrolytes and on the other hand, the adrenal medulla and its constituents are essential for the maintenance of the intrinsic properties of the heart and to a large extent the tone and responsiveness of the peripheral vascular system. The final common denominator of the peripheral vascular system is the capillary unit, which controls the ability of the organism to carry on the necessary metabolic functions of life—those of internal respiration, nutrition and elimination.

It is the role of the secretions of the adrenal medulla and some of the anesthetic problems in their management, which I wish to emphasize.

We are all aware of and have seen the tragedies of the admixing of too high a circulating blood level of epinephrine in the induction of anesthesia, especially with cyclopropane or ethyl chloride. I refer, particularly, to the frightened child inadequately premedicated, who is induced under general anesthesia with such a high blood level of circulating epinephrine that ventricular fibrillation and cardiac arrest are the not uncommon outcome. We have all learned that too high a circulating epinephrine level can be wrought with these hazards and it is for this reason that we have stressed the adequate sedation of patients presented for anesthesia. We are all aware also, by virtue of the magnificent contributions of Brewster and Isaacs, that we must have at least a minimal concentration of epinephrine available in the blood stream in order to maintain normal blood pressure, under the influence of ether anesthesia. However, very few of us consider the great scope that epinephrine plays in other anesthetic managements, and I think that we should point our interest in this direction for the remainder of this discussion.

The basic chemical compounds of the adrenal medulla and the active principals are chemically classified as catecholamines—substances which include both epinephrine and norepinephrine.

Of significance in the pharmacologic understanding of the actions of these catecholamines is the fact that as well as being present in the adrenal medulla, they are also stored in other chromaffin substances of the body so that they may be immediately called upon to support the organism in the so-called "fight or flight" mechanism. The second important consideration is the work of Suchian Ngai and Jack Frumin at the College of Physicians and Surgeons, who have demonstrated that these compounds function at the capillary unit by stimulating recep-

tors in the effector cells. They have demonstrated in dogs that upon stimulation these receptor cells will produce both vasoconstriction and vasodilatation which would correspond to the original concept of E and I substances considered to be active in epinephrine activity as projected by Cannon and his workers many years ago. The E receptors are stimulated by both norepinephrine and epinephrine. The I receptors are stimulated only by epinephrine.

It is, therefore, permissible to project that the outcome of norepinephrine secretion and stimulation is only vasoconstrictor response. It is equally justifiable to postulate that epinephrine secretion will result in either vasoconstriction or vasodilatation, but in the presence of adrenergic blocking agents such as Regitine, the E receptors would be blocked from both epinephrine and norepinephrine stimulation of these receptors. Adrenergic blocking agents will not inhibit the responsiveness of I receptors which would result in marked vasodilatation in the presence of adrenergic blockers and epinephrine secretion. The result of such a situation would be disastrous hypotension.

The clinical application of these facts is most fascinating in the anesthesiologic management of two clinical types of cases. In the first category with the description which we have rendered of the dependence of the body on substantial epinephrine stores in the chromaffin tissues, we are seeing more and more hypotension in patients who have been on hypotensive drug therapy. These drugs include many pharmacologic categories. The phenothiazines have both central nervous system and ganglionic blocking activity in producing hypotension and there are those known as the rauwolfia alkaloids, such as Serpasil, Hydropress, Rauduxin, etc., which produce their response by inhibiting the production of epinephrine and norepinephrine in chromaffin tissues. It has been demonstrated in dogs that a dose of 0.4 milligrams per kilo is able to produce this type of response. This has not been correlated for human subjects. The depressant activity of rauwolfia may remain for 10 days-2 weeks.

Now it is recognized that adequate circulating epinephrine is essential for the maintenance of normal blood pressure under certain of the major general anesthetics that are employed, for example, ether. Many clinics, including our own, have found disastrous hypotensive crises in patients who have been subjected to both the medical management of their hypotensive states and the application of any of the general anesthetics that are in common use as well as regional anesthetics, such as spinal and epidural, when the anesthetic has been employed either during rauwolfia alkaloid therapy or within a 2-week period following the cessation of such therapy. Subsequent to this 2-week withdrawal phase there has been no severe synergism in the production of difficult or unmanageable hypotension. The hypotension created by the combination of the rauwolfias and anesthetics has only been responsive in our hands to the administration of large doses of norepinephrine—a truly heroic and potentially dangerous management of an avoidable situation in elective surgery.

When the surgery has been deemed emergent to the life of a patient, these patients have been undertaken for anesthesia with the employment of anesthetic agents which would not be dependent upon concentrations of cer-

tain catecholamines for maintenance of blood pressure. Simultaneous administration of norepinephrine infusions throughout the surgical or postoperative experience has been effected —until they have been satisfactorily weaned from the general organism depression of the anesthetic agents.

The second major group which we have to consider are those patients in whom there is concern for catecholamine over-secretion. These are best exemplified by the patient suffering with pheochromocytoma who is presented for surgery and anesthesia. Since from 80% to 85% of such patients would be producing norepinephrine rather than epinephrine, the potential threat of disastrous hypotension during and postanesthesia is not of too much concern following the employment of Regitine block for any hypertensive crisis that might ensue by simply inducing anesthesia or by surgical manipulation of the tumorous gland. In norepinephrine secreting pheochromocytomas, it is a common clinical experience to observe the restoration of blood pressure to normal manageable levels following Regitine block.

The truly difficult patient to manage with pheochromocytoma is the one who secretes epinephrine from his tumor. In these patients, immediately following Regitine block, the blood pressure is often restored to reasonable levels. With further surgical manipulation of the gland and a resultant increase in circulating epinephrine, the disastrous hypotension occurs with the stimulation of the I receptors by the excess epinephrine. The I receptors at this point, have not been blocked by Regitine which has, however, blockaded the activity of the epinephrine on the E receptors. The result is an unopposed vasodilatory response. Further administrations of either epinephrine or norepinephrine may indeed make the situation worse. It is here that the surgeon must withdraw and the anesthetist must pray—for the situation is truly in the hands of God alone.

A provocative consideration to prevent such episodes from reaching their full climax in this type of patient is to produce an incomplete Regitine block at the time of any initial hypertensive crisis until the tumor is pharmacologically diagnosed at the table. Should marked hypotension occur with glandular manipulation, the E receptors will not be completely blocked and will remain somewhat responsive to the administration of norepinephrine as a therapeutic tool.

SUMMARY

In summary then, we have today pointed up our greatest statistical complication. We have not belabored you with the things you already know so well, but we have specified that there is still a great deal to consider in hypotensive patients whose states might have been created by an oversight in the consideration of the adrenal medullary function. We have marked a most fascinating experience with the I and E receptors in pheochromocytoma and, in addition, we have stressed the ever increasing hypotensive response with the patient who is on medical management of hypotension with the rauwolfia alkaloids and who is exposed to an anesthetic experience while chromaffin tissues are still depressed. It is here that many situations of hypotension can be avoided by a thorough understanding on the part of the internist, the surgeon and the anesthetist, of the

(Continued on page 106)

Pre-operative Evaluation and Treatment of Pulmonary Disease

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Abnormal pulmonary function constitutes one of the greatest hazards to safe anesthesia.[1] In most cases, this hazard lies in the anesthetist not being aware of the abnormality until after induction of anesthesia. At this point, the anesthetist is trapped in a situation from which he may not be able to extricate himself or the patient.

In the preoperative patient evaluation, the best laboratory test to assess pulmonary function is time, thought and energy spent with the patient prior to surgery. It is extremely difficult and often impossible to make up during and after surgery what has not been done and should have been done before surgery. In eliciting the past medical history such questions as the following should be asked:

1. Have you had previous lung, nose, or throat infection such as pneumonia, bronchitis, tonsillitis, or sinusitis?
2. Have you ever had lung or chest wall surgery? Previous surgery and anesthesia of any type? Were any difficulties encountered?
3. What type of work do you do? Where do you work? Have you ever worked in coal or other mines? In steel or other dusty occupations? As a glass blower? (Pneumoconiosis, silicosis)
4. Have you ever resided in the West or South? (Coccidiomycosis and Boeck's Sarcoidosis)
5. How much do you smoke? Here observation of the patient's bedside stand may be informative.
6. Family history of lung disease. (Tuberculosis, emphysema)

The functional inquiry should include such questions as to determine the presence of cough, amount and color of sputum and when produced, exertional dyspnea, orthopnea, wheezing and chest pain. For some reason patients seem reluctant to admit to abnormalities of breathing so that the anesthetist often has to elicit information in an oblique manner. In male patients, a question about hunting or fishing activities and in female patients questions about household activities, gardening, or such sports as bowling will give some idea as to actual functional cardiorespiratory ability.

In the physical examination, chest size and shape are important in excluding emphysema and spinal deformities. Pleural fluid, retained secretions, consolidated areas, large lung cavities and air flow obstruction may be determined by percussion and auscultation.

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Two simple tests may be made during the physical examination which will give a reasonably accurate estimation of the patient's functional cardiorespiratory ability. The first is to have the patient walk up one flight of stairs with you. Comparison of before and after pulse and respiratory rate with your own will tell you whether the patient has more or less cardiorespiratory reserve than yourself. Of course, this requires that you have some idea of your own reserve. The second test is called the match test.[2] The patient is requested to blow out an ordinary lighted book match held six inches from the widely opened mouth. If the patient is unable to do this, a defect in air flow is present and requires further investigation.

Evaluation of the patient up to this point has required probably not more than 10-15 minutes, has not required any special gadgets or cost the patient any money. Only an occasional patient will require further examination in the form of the following six laboratory tests:

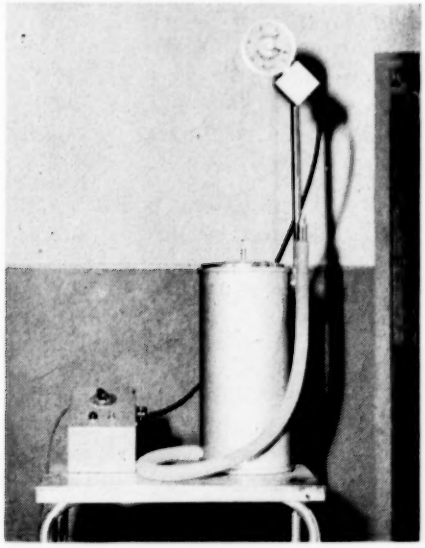

Fig. 1 Collins Ventilometer

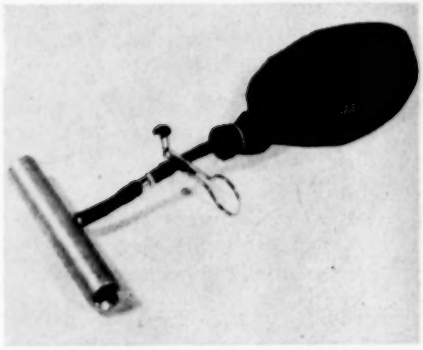

Fig. 2 Warring-Siemsen VENTUBE for measuring maximum breathing capacity and exercise ventilation.

1. AP and Lateral X-ray of the chest. Particular attention should be paid to the presence or absence of emphysema, fibrosis, pleural fluid or air: rib and spinal deformities, size and shape of the cardiac and aortic shadows, parenchymatous masses or cavities, and substernal thyroid enlargement.

2. Timed Vital Capacity. (Collins Ventilometer, Fig. 1.) The normal vital capacity usually is indicative of a restrictive defect such as is seen with thoracoplasty. In the Timed Vital Capacity, the amount of air exhaled is determined per unit of time, usually one, two or three seconds, the most important of which is the first second. A normal one second T.V.C. is at least 75% of the total vital capacity. A reduction below 75% indicates an obstructive defect such as emphysema.[3]

3. Maximum Breathing Capacity is exactly as termed. (Warring-Siemson Tube, Fig. 2.) The M.B.C. may be reduced when the V.C. is normal and gives

a more accurate estimation of the patient's total cardiopulmonary status.

4. Pulmonary Spirometry correlates and gives a permanent record of the inspiratory capacity, expiratory reserve volume, tidal volume, oxygen uptake, and minute ventilation. (Collins Double Spirometer, Fig. 3.)

5. Arterial CO_2 tension can be determined simply by obtaining end alveolar carbon dioxide tension (Fig. 4), and is elevated in such diseases as pulmonary emphysema, and long standing asthmatic bronchitis.

6. Arterial oxygen saturation may be determined by ear lobe oximetry. Cyanosis is often not readily apparent until arterial oxygen saturation is below 85%. Oximetry is particularly valuable in determining small degrees of arterial unsaturation.

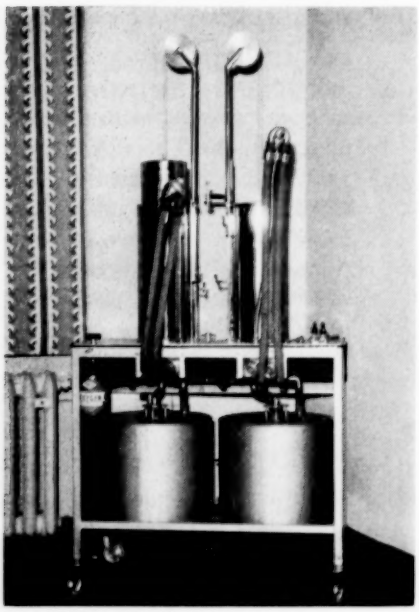

Fig. 3 Collins Double Spirometer

Fig. 4 Collins End-Alveolar Carbon Dioxide Tensionometer

PRE-OPERATIVE THERAPY

Determination of an abnormal lung function in your patient increases your chances of a successful anesthetic as you will not now walk into the case fat, dumb, and happy. You will give more consideration to the type of anesthesia, drugs to be used and care in technic. Improvement in pulmonary physiology can be obtained in most patients.

1. Asthma: Bronchodilators and vasoconstrictors such as Isuprel®, epinephrine, and aminophylline may be combined with mild sedatives to produce improvement. Endobronchial infection requires sputum culture and sensitivity determination and then proper antibiotic therapy. Intermittent positive pressure inhalations may be helpful.

® Winthrop trade name for isoproterenol hydrochloride.

Steroid therapy is usually reserved for severe, resistant cases which may also require treatment of right heart failure.

2. Chronic bronchitis (smoker's bronchitis) will respond to a program of reduced or abstinence of smoking, sputum culture and sensitivity with appropriate antibiotics, intermittent positive pressure with Alevaire® or other wetting agents.

3. Hydrothorax, pneumothorax, active tuberculosis, unexplained pulmonary masses or cavities, require appropriate investigation and therapy. Only acute surgical emergencies are handled in this situation. Pleural fluid can be partially drained prior to induction of anesthesia. X-ray should be repeated as a pneumothorax may be induced while aspirating pleural fluid. Pneumothorax should be treated with under-water catheter drainage.

4. Bronchiectasis may be suspected if the patient states that most of his coughing and sputum are produced in the morning shortly after arising from sleep. He often has a history of recurrent pneumonitis and pleurisy. Hemoptysis, when it occurs, frightens the patient into having proper investigation. Effective preparation for surgery is essential and consists of supervised postural drainage, aerosols and bacteriologically controlled antibiotic therapy.

5. Emphysema may be separated into two distinct forms: the elderly patient with the emphysematous type rib cage but with few or no functional symptoms does not require investigation or therapy. However, the destructive type of emphysema associated with recurrent acute or subacute bronchitis, bronchospasm, and marked loss of alveolar and vascular lung tissue is a serious problem. Varying degrees of carbon dioxide retention, arterial oxygen unsaturation, elevation of pulmonary arterial pressure and right heart failure exist. Pneumonitis, endobronchial infection and edema, bronchial collapse, chronic severe fatigue and malnutrition co-exist. Surgery and anesthesia in this type patient must never be approached lightly. In my experience, severe forms of this disease are the most difficult to treat and are a nightmare to the anesthetist. For a complete discussion of emphysema, the Symposium in the March 1958 issue of the British Journal of Anaesthesia is excellent.[5]

It is often perplexing to decide where to start in the treatment of people with emphysema. Aerosol bronchodilators and vasoconstrictors with wetting agents are a beginning point. Mild sedation is often helpful. Digitalization for right heart failure is introduced but often is not particularly effective until some correction of the abnormal arterial carbon dioxide and oxygen values is obtained. Intermittent positive pressure inhalations with the previously mentioned aerosols and 40% oxygen may be used with restraint on the amount of positive pressure. Tracheostomy decreases dead space and aids in the removal of secretions, and it may be of great benefit in the postoperative period. Death from tracheostomy in emphysematous patients has been re-

® Winthrop trade name for detergent.

ported and is most likely related to sudden changes in arterial carbon dioxide tension or bilateral tension pneumothorax.

Diamox®, by producing bicarbonate diuresis, may decrease the respiratory acidosis.[6] However, Diamox is a two-edged sword by producing metabolic acidosis. Recently, a new drug, Tris (hydroxy methyl) Amino Methane, abbreviated to T.H.A.M., is being evaluated in the treatment of respiratory acidosis. Briefly T.H.A.M. corrects the arterial pH toward normal, promotes diuresis, increases arterial oxygen saturation and seems to increase the effectiveness of digitalization. Hypoglycemia has been reported as a side effect of the drug.[7] More reports on this drug are eagerly awaited.

® Lederle brand name for acetazolamide.

SUMMARY

Preoperative evaluation of pulmonary function can be obtained by an adequate history, functional inquiry and a few simple tests. A few days of treatment before surgery may prevent serious anesthetic and surgical complications.

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Recognition and Treatment of Cardiac Emergencies Under Anesthesia

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Real cardiac emergencies under anesthesia are not rare and they all too often occur when least expected. It has been well documented that the catastrophes under anesthesia are relatively more common in routine operations on patients preoperatively considered satisfactory risks. With the increasing scope of surgery to include massive resections, formidable intracranial operations, cardiac surgery, etc., under more complicated and hazardous conditions such as hypotension, hypothermia and extracorporeal circulation, the number of operating room deaths must, at least for a time, increase. This should obviously not deter us from attempting to push forward the frontiers of surgery and anesthesia since we are helping or curing many patients who otherwise would suffer or succumb to their disease or congenital malformation.

We are going to consider chiefly the recognition and treatment of cardiac emergencies that arise during routine average general hospital procedures. These are the heartbreaking, frequently unexplainable, disasters that constantly urge and demand that the anesthetist carefully evaluate his patient and give him the complete undivided attention that is required of the anesthetist and owed to the patient. There is no existing practical battery of monitors or other mechanical devices that compares with the careful intelligent evaluation made by the observations of a well trained anesthetist. Many of the monitoring devices can be of real aid but when they are relied upon to any degree that will distract us from unremitting vigilance they are fraught with disaster. The constant "beeping" or flashing of a pulse or "R" wave monitor may lull one into a false sense of security that allows him to be distracted from the far more informative palpation of the peripheral pulse. We will not labor over the advantages or disadvantages of these devices except to re-emphasize that they are only aids and they in no way eliminate the constant surveillance demanded of the anesthetist.

It is probably a fact that most deaths attributable to anesthesia or better still, to the anesthetist, are preventable. We do not mean to infer that they are due to negligence, but are rather due to a combination of perhaps unknown and frequently unrelated circumstances that occur during the administration of the most potent and lethal drugs employed in medicine. It has been said that the

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anesthetist carries a patient to death's door and back. Occasionally one or more circumstances prevail that cause this door to be left open and the unfortunate anesthetist carries him through it to the great beyond.

In considering cardiac emergencies we must not allow ourselves to think only of mortalities, because these are rare. More often we are concerned with morbidity and this can be as tragic as a death. The patient who survives his immediate surgery and anesthesia and is allowed the privilege of suffering through a painful convalescence, only to be faced with coronary occlusion or insufficiency that ends in heart failure and death, is not a great deal better than one who succumbs during the peace of narcosis.

Morbidity is extremely difficult to evaluate, but it is important and our attention must be attracted to it and we are urged to find ways of statistically evaluating results so that they can be properly appraised. This will require complete preoperative evaluation, accurate detailed anesthesia records which include pertinent notes referring to events during surgery, such as traction reflexes, acute hemorrhage, change in position, insertion of packs, etc., and a careful, honest postoperative follow-up. Even with all of this information—usually unavailable on hospital charts—a group of unbiased specialists could only make an educated guess as to what really is responsible for postoperative morbidity.

The heart ceases to function because the myocardial fibers receive insufficient oxygen or they are chemically rendered incapable of contracting or relaxing. This must be true because the heart has "auto rhythmicity" which allows it to beat without outside stimuli. Anoxia is by far the commonest factor and it may be due to any of the well known types of anoxia—anoxic, anemic, stagnant or histotoxic. Cells are rendered incapable of contracting by chemical changes which effect the fibers themselves or the polarity and permeability of the cell membrane which may paralyze them. Examples of this latter group are such things as severe acidosis, excessive potassium or calcium, profound anesthesia, etc. Only by recognizing the clinical and laboratory findings which indicate early manifestations of any of these unfavorable influences and the immediate institution of corrective measures can we hope to avoid catastrophies in anesthesia.

They may arise when we anticipate them, but they all too often occur insidiously or without warning, and with alarming rapidity cardiac function ceases. Only with constant observation of the respiratory pattern, color of the blood, rate, rhythm and volume of the pulse, the blood pressure and the condition of the skin and peripheral blood vessels, can we hope to make anesthesia safe and allow patients to enjoy the benefits afforded by surgery. These observations cannot be made or intelligently treated if the patient has not been adequately evaluated preoperatively by a complete history and physical examination and the necessary laboratory studies properly interpreted.

Premedication must be individualized and its effect should be established and evaluated before any anesthetic is begun. An extremely hazardous practice of giving these drugs on call to the operating room is seen much too frequently. This is especially true in busy hospitals where large volume renders us callous and

we neglect the fact that this morning's "gall bladder" is really a frightened soul who has never before been in a hospital. The fact that she had been awakened by a noisy nurse who stuck a thermometer in her mouth, shoved an enema tube in her rectum and was urged to hurry in expelling it because the O.R. was waiting did little to calm her fears and much to stimulate epinephrine secretion.

When the anesthetist greets the patient she must be kind, sympathetic, reassuring and unhurried. A smile and a soft voice that confirms identity and explains what is to be done will earn its just reward. During this time the conscious breathing pattern, condition of the skin, color of nail beds and size of pupils should be observed.

Induction ideally is smooth and the depressant anesthetic drugs given slowly so that compensatory mechanisms can manifest themselves and the patient's tolerance carefully appraised. This is the most hazardous anesthetic period and the anesthetist should have an informed assistant to aid him at least until the anesthesia is established. During this period the pulse should be constantly palpated and this is often impossible for one person who is maintaining an airway and adjusting flow meters.

Cardiac arrest upon insertion or manipulation of an endotracheal tube occurs all too frequently and we have a strong feeling that many factors act in concert to produce this tragedy. The much maligned "vagal-vagal" reflex may play a part, but we feel increased endogenously secreted adrenalin, drug depression, hypoxia and hypercarbia set the stage for it and by preventing them the reflex would only rarely manifest itself. The visceral reflexes that induce cardiac arrest are, likewise, seen much more commonly during periods of hypoxia and hypercarbia. To avoid this we must ask the surgeon to discontinue the stimulation of these reflexes until these conditions are corrected. Surgeon and anesthetist must be mutually helpful, considerate and generous if they are to avoid calamity.

Anoxia from any cause is undoubtedly the commonest cause of cardiac arrest. Allow us to outline the types of anoxia and list the most frequently encountered causes during anesthesia. By early recognition and correction of them many emergencies will be prevented.

TYPES OF ANOXIA

I. **Anoxic anoxia**—where arterial blood is incompletely saturated.
   a. Inadequate tidal exchange due to depression from drugs, obstruction of the airway, muscle paralysis, position, packs, pneumothorax, improperly placed endotracheal tube, etc.
   b. Inhalation of less than twenty per cent oxygen due to attempts to employ weak anesthetics such as unsupplemented nitrous oxide; faulty adjusting of flow meters; improperly calibrated gas machines; unrecognized decrease in tank pressure which causes the flow rate to fall; tanks erroneously filled or connected to the gas machine.
   c. Interference with exchange through the alveolar capillary membranes as seen in edema that follows aspiration of gastric contents; edema due to heart failure, etc.
   d. Insufficient functioning lung tissue.

II. **Anemic anoxia**—the result of inadequate amounts of oxyhemoglobin being delivered to the tissues secondary to anemia. This may be the result of hemorrhage or a true anemia.

III. **Stagnant anoxia** results from sluggish blood flow due to myocardial failure, peripheral vascular collapse, low blood volume or reduced return flow to the heart. This form of anoxia is very often seen in the anesthetized patient and will be referred to later.

IV. **Histotoxic anoxia** is seen in cyanide poisoning and perhaps after large doses of barbiturates, narcotics and profound anesthesia.

May we now consider the untoward clinical findings that regularly confront the anesthetist and outline a program that should correct them.

Hypotension is a serious complication and its cause must be immediately determined and corrected by appropriate means. This does not imply that every hypotensive patient should receive a pressor drug. This error is made too frequently and may do more harm than good. Under most general anesthetics the peripheral vascular bed is increased by vasodilatation and the myocardial efficiency is reduced. As anesthesia proceeds to deeper planes this process may progress to produce stagnant anoxia of the heart as well as such important organs as the brain, liver and kidneys. Moderate hypotension for relatively short periods of time in healthy patients is well tolerated, but in the cardiac patient or a patient with liver disease this may lead to irreparable damage.

In the patient with myocardial disease even short periods of hypotension may suddenly lead to disaster. If the disease is due to coronary narrowing, a high head of pressure is needed to allow sufficient blood to pass through the vessels. They are frequently sclerotic and incapable of dilating as normal coronary vessels do in the presence of hypoxia. The failing heart will quit when its work load is increased, its fuel supply diminished and the products of metabolism are allowed to accumulate. Much is being learned about electrolytic exchange, membrane permeability and polarity which perhaps will eventually afford us a means of more direct regulation of the function of cells.

Hypotension must be corrected by eliminating its cause or providing a pharmacologic antagonist to aid in restoring normal physiology. When this occurs under inhalation anesthesia the bag should be emptied and refilled several times with oxygen. Respirations may be supplemented to "feel" if there is any increased resistance and if the patient is under controlled respirations we should try to establish spontaneous breathing to take advantage of the thoracic pump. All other possible causes should be investigated including the removal of packs and retractors to eliminate sources of reflex stimulation and more commonly relieve vena caval obstruction or hidden hemorrhage. Here again, the surgeon must be apprised of the situation and we seek his indulgence until the patient is in satisfactory condition. Occasionally, and perhaps increasingly with the use of muscle relaxants, a patient will be too light and it might be necessary to deepen the plane of anesthesia. When in doubt we prefer to awaken the patient enough to make him move or perhaps cough from the irritation of the endotracheal tube. We have no explanation for it, but frequently

after a patient coughs on the tube his pressure returns to suitable levels.

Obviously, blood loss should be treated by blood transfusion and blood substitutes. Pressor drugs are only useful until blood can be replaced.

We almost never use a vasopressor during general anesthesia until other possible factors are eliminated. When we are forced to use one we have found that twenty-five (25) milligrams of ephedrine given intravenously does an excellent job. We use it because it is a vasoconstrictor as well as a myocardial stimulant. If hypotension persists other drugs must be used.

Under spinal or epidural anesthesia the fall in pressure is due to decreased return flow to the heart secondary to peripheral vasodilatation and pooling of blood in the increased vascular bed. Intravenous pressor drugs are indicated and because they are frequently needed immediately it is probably a good rule to start an intravenous infusion before each spinal or caudal anesthetic is given. An increase in blood pressure from 50 mm. Hg. to 150 mm. Hg. at the mouth of the coronary increases blood flow ten fold.

When hypotension is due to myocardial failure, rapid digitalization and abandoning of the procedure is recommended unless there is real immediate benefit to be derived from it.

Reviewing the physiology of the heart will demonstrate the urgency of immediate therapy. From Starling's law of the heart we know that the better the heart fills the longer will the myocardial fibers be and the more forceful will be the contraction. With peripheral resistance remaining constant, the more forceful contraction will increase the blood pressure and through the carotid and aortic sinuses will slow the heart. Slowing the heart rate allows a longer diastolic pause which results in better filling. By better filling the heart can increase its output 300%. Another often forgotten benefit of a slow rate is that flow through the myocardium is interferred with by contraction of the muscle so that a longer diastole encourages better perfusion of the heart. Finally the heart uses oxygen during contractions and the less frequently it beats the less will be its oxygen requirement.

Hypoxia affects the myocardium in a very similar vicious cycle. The carotid body increases the heart rate which increases the work of the heart against increased peripheral resistance. The fibers perfused with poorly oxygenated blood do not contract well and, therefore, the heart becomes a less efficient pump. The arrhythmias favored by hypoxia may further challenge the heart.

Hypercarbia of itself probably only increases the rate, work and sensitivity to arrhythmias; but when it progresses to acidosis the membrane potential, cell function and electrolytic balance of the myocardial fibers is severely deranged to compound the pathologic process.

Arrhythmias during anesthesia are frequently due to the effects of drugs on conduction through the myocardium and to hypoxia and hypercarbia. There is adequate evidence that epinephrine plays a role in many of these arrhythmias since they are prevented by dibenamine. It is also noteworthy that they are less common following a Pentothal induction. Arrhythmias are frequently encountered in patients with pre-existing heart disease, especially when they are on digitalis. Ectopic beats are often initiated by

stimulation of abdominal viscera, the pericardium, pleura or hilum of the lung and they are believed to be due to vagal stimulation. Large doses of atropine are said to decrease their sensitivity, but it may cause annoying tachycardia. In patients with partial heart block atropine is helpful, but in complete block it has less value. Isuprel and epinephrine are the drugs of choice in the Adams-Stokes syndrome.

Sinus tachycardia can frequently be slowed by vagal pressure which helps us to differentiate it from the much more serious ventricular tachycardias which may be just one step before ventricular fibrillation and, therefore, demands immediate treatment. As we have indicated earlier, any tachycardia increases the work of the heart and interferes with its efficiency.

As a general rule the supra-ventricular arrhythmias are less serious, but they do indicate a change in conduction or stimulus and attempts should be made to correct them. We find that lightening the anesthetic is helpful in most of these and we often change to another drug if they persist. Many preoperative drugs have been suggested that would eliminate these changes in rhythm, but they have not been very successful. Procaine is of value in ventricular arrhythmias, but it is a myocardial depressant and should be used only when specifically indicated. Quinidine is a vagal depressant and is of value chiefly in ventricular arrhythmias, but it is a moderately severe myocardial depressant and it may increase the ventricular rate and precipitate heart failure. Digitalis is of value in the treatment of congestive failure and certain ectopic auricular rhythms.

The heart that stops beating is likely to remain stopped unless it is stimulated directly. Intravenously administered drugs are of no value since they will not reach the heart even if they were therapeutic. When one suspects that circulation has ceased he is obligated to inform the surgeon of the situation so that he can confirm it and immediately institute resuscitative measures. In this situation time is a priceless commodity and it cannot be wasted. It would be better to open a chest prematurely to find the heart beating feebly rather than spend time confirming the diagnosis. In a patient who suddenly becomes pulseless, we have no right to seek other evidence of cardiac arrest. The absence of pulse is confirmed quite rapidly by the anesthetist failing to feel a radial, carotid or femoral pulse. The surgeon should palpate the aorta or any easily accessible large artery. If a feeble pulse is present, a vasopressor such as neosynephrine may be given intravenously, the table put in steep Trendelenburg position, and all packs and retractors removed while the lungs are being inflated with fresh supplies of oxygen. Epinephrine is probably a dangerous drug in this situation since it might throw the hypoxic myocardium into ventricular fibrillation. It has been suggested that in this emergency, epinephrine be given with fifty to one hundred milligrams of procaine to avoid fibrillation.

As soon as cardiac arrest is suspected time should be measured and everyone should be alerted to its passing at thirty second intervals. This is important since the magic four minute interval passes all too soon. Sudden absence of pulse demands cardiac resuscitation. There is no time for listening for the heart sounds,

getting E.K.G. tracings or looking at retinal vessels.

Two emergencies exist—one of lack of circulation and the second of apnea. Apnea is treated by artificial respiration with 100% oxygen via an anesthesia machine bag and mask. When convenient, an endotracheal tube may be inserted to facilitate this and to avoid distension of the stomach.

If open cardiac massage is to be used, the chest should be entered through the fourth interspace on the left, and cartilage of the fourth and fifth ribs cut near the sternum. This allows us to see the heart and begin massage. As soon as adequate massage is begun the emergency is over because vital tissues are being oxygenated. Massage should produce a palpable carotid or femoral pulse and frequently a recordable blood pressure. If the heart is in asystole or fibrillation, massage should be continued for at least five minutes or until spontaneous beating starts. By this time rib spreaders and instruments should be available to open the pericardial sac for easier manipulation.

For fibrillation, short shocks of 1.5 to 2 amperes for one-half second are most efficient. If one shock fails, the heart should be massaged again and then given three rapid shocks at one second intervals. If this fails, 70 mgm. of procaine may be injected into the left ventricle and, after forcing it through the coronary vessels, the shocks re-applied.

For asystole that fails to respond to massage a solution of 0.5 cc. of epinephrine in seven cc. of 1% procaine may be injected into the chamber of the left ventricle and massage continued. Another drug that may be useful at this time or if very feeble contractions are present is the injection of 4 to 6 cc. of 10% calcium chloride. Clamping of the descending aorta will aid in perfusion of the brain, heart and lungs. Massage should be carried out for at least two hours before all efforts are discontinued.

Here again, return flow to the heart is important and one should employ steep Trendelenburg position and rhythmic pressure on the abdomen to force blood into the heart.

Our experience with pacemakers has been disappointing, but there may be a place for this instrument.

When spontaneous beats have begun and circulation is adequate the chest should be allowed to remain open until the operation is completed. Needles to say, only emergency surgery should be completed at this time.

Hypothermia has proven to be of value in preventing cerebral edema and hyper-pyrexia and apparently increases the survival rate.

SUMMARY

Cardiac emergencies are most frequently secondary to hypoxia, hypercarbia, viserocardiac reflexes and drug overdosage. These can only be avoided by constant, intelligent observations on the part of the anesthetist. Some operating room mortalities can be prevented by close cooperation between the members of the operating team and the anesthetist. The essentials of open cardiac massage are described.

Since this paper was prepared an excellent method of closed cardiac resuscitation has been described and everyone in medicine should acquaint themselves with this efficient and less hazardous technique.

Notes and Case Reports

Endotracheal Tube Holder

All anesthetists have, at one time or another, been forced to use a stylet to facilitate endotracheal intubation because of a limp, poorly curved tube. The stylet complicates the intubation, prolongs instrumentation and is potentially traumatic.

Gillespie, in his text, "Endotracheal Anesthesia," suggests the use of a round basin in which to store the endotracheal tubes, thus maintaining the proper curvature. While that method is effective, the tubes must be stacked one over the other, making access difficult. Presented below is a simple device for storing and maintaining curvature of plastic endotracheal tubes. Constructed from scrap wood and ordinarily discarded plastic parts, the cost should not exceed one dollar. Built as described, the device will hold eight endotracheal tubes, sizes: French 26-40.

Materials:

One 8 inch x 5 inch piece of 3/8 inch plywood
Eight 1 inch, #14 flat head wood screws
Eight 3/4 inch, #10 flat head wood screws
Disposable covers from eight Abbott Blood Administration sets, #4436 (Figure 1). (Undoubtedly, these sets can be garnered from similar sets of other manufacturers with modifications.)

a. plastic cannula covers—8
b. plastic airway needle covers—8
c. plastic needle covers—8

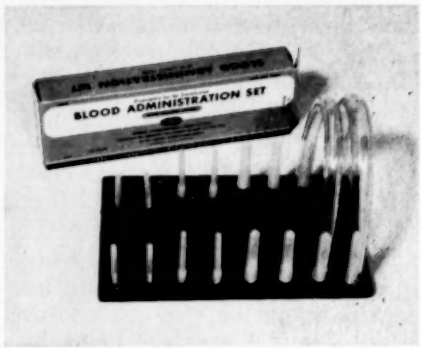

Figure 1

Construction:

1. Drill plywood and counter-sink holes as illustrated in Figure 2. The first four pair of mounting holes are 1/4 inch in diameter; the remaining four pairs of holes are 3/16 inch in diameter.

2. Sand and varnish, or paint as desired. When completely dry, spray with three coats of Krylon® Crystal Clear plastic spray. The plastic coating results in a durable, easily cleaned surface.

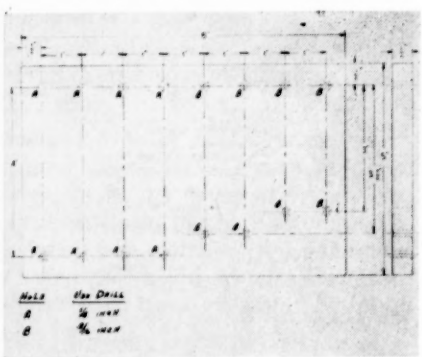

Figure 2

3. Insert the one inch screws through the four pairs of holes that are four inches apart. Screw the plastic air filters onto each screw after removing the cotton pledget. The fit will be quite tight, spreading the filters widely.

4. Using hot water, soften the plastic cannula covers and force each into place over the air filters. When cool, the cannula covers will be very difficult to remove.

5. Insert the 3/4 inch screws into the remaining holes.

6. Attach the plastic needle covers directly onto the two pair of screws that are 3-1/2 inches apart.

7. Remove the wide base of the remaining four needle covers and attach to the last two pair of screws that are 3 inches apart.

When preparing the endotracheal tubes for the initial insertion on the holder, soften them by soaking in hot water for a few minutes. Place the tubes on the holder while still pliable and allow to cool in position, preferably overnight, thereafter attach immediately following washing. When removed, the tubes will seem to have assumed too great an arc. However, they will straighten slightly in the interval between removal and actual intubation.

In the two years that the tube holder has been in use at the Polk County Hospital, we have seldom needed the assistance of a stylet for intubations, even though we routinely use the Macintosh Laryngoscope blade which is reported to require endotracheal tubes of greater curvature.

Robert J. Higgins, C.R.N.A.
Bartow, Florida

ANNUAL MEETING
1961
ATLANTIC CITY
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September 25-28

Combined meeting of school directors and state officers, Sunday, September 24th.

Watch for your hotel reservation forms to be mailed this month.

The THIRTY-THIRD QUALIFYING EXAMINATION for membership in the American Association of Nurse Anesthetists will be conducted on May 13, 1961. The deadline for accepting completed applications including the transcripts is April 1. Notice of eligibility will be mailed about April 10.

Applications should be forwarded early enough to allow time to request transcripts and have them returned to the Executive Office before the deadline date.

The Fall Examination will be held on November 11, 1961. Applications and transcripts should be sent before November 1.

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(For further information see page 95.)

John C. Maginnis

Insurance Consultant

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Plan 4 —	50,000/ 150,000	10,000	250	17.00	45.90
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(Read the article on page 94 for full particulars.)

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Legislation

Emanuel Hayt, LL.B., Counsel A.A.N.A.

Failure to Maintain Proper Flow of Oxygen Not Proved to be Cause of Death

Plaintiff brought suit to recover for the wrongful death, pain and suffering and burial expenses of her decedent, allegedly resulting from the failure of defendant's hospital to maintain the proper flow of oxygen to decedent.

The nurses and aids denied any trouble in changing from one tank to another or any unnecessary delay in restoring oxygen to the patient when the changes were made. Testimony by the nurse and aid on duty on the morning patient died testified at the time they changed tanks at two o'clock, there was 400 pounds pressure on the tank then being used but the change was made to suit the patient; that patient was having a hard time breathing and after the change was made he threw off the mask and complained he was not getting any oxygen; that they changed back to the old tank and used the nose catheter and he then was cyanotic, pulse very low and he was gasping for breath and the nurse gave him another injection to stimulate the heart.

No testimony was produced by the plaintiff to the effect that the lack of oxygen as above outlined or the commotion complained of was the cause of the death.

In an action for the recovery of damages for wrongful death of decedent, the burden of proof is upon the plaintiff to establish by competent evidence that a negligent or wrongful act of defendant was the proximate cause of death. Plaintiff did not establish by competent evidence a causal connection between the alleged acts of negligence of defendant and the injury to the decedent or that the alleged acts were the proximate cause of his death.

(Jinx v. City of Norman, 11 CCH Neg. Cases 2d 224-Okla.)

Paralysis of Patient After Anesthetic Injection Not Proved to be Caused by Negligence

This is an action to recover damages for personal injuries. The plaintiff was a patient of the defendant, a practicing physician, and claims his injuries are due to negligence. The claim of malpractice is based upon the administration by the defendant of an anesthetic. At the close of the plaintiff's case the defendant moved for an involuntary nonsuit, which was granted, and the plaintiff appeals.

The record further discloses that in administering the anesthetic in question the defendant's object was to anesthetize the areas supplied by the motor and sensory nerves stemming from the sacral and lumbar regions (the lower portion of the back). The desired result may be accomplished either by administering an anesthetic in the sacral canal, which is located between the coccyx (tail-

bone) and the subarachnoid space, or by administering an anesthetic in the subarachnoid space, which contains the spinal cord. The subarachnoid space begins a few inches above the coccyx and continues up the backbone toward the head. The former procedure is called a "caudal block"; the latter a "spinal." Different strengths and types of drugs are used for each. The defendant stated that he intended to and did administer a caudal block in the instant case. The use of the caudal block is good medical practice. A caudal block anesthetic is to operate "extra dural," that is, it is not intended that the injected fluid should enter the subarachnoid space.

A doctor also testified that in his opinion the reaction suffered by the plaintiff was due to the "idiosyncrasy" of the plaintiff's physical makeup and not to an injection of the anesthetic into the subarachnoid cavity. No other expert testimony was offered by the plaintiff. The defendant was called as an adverse witness and his testimony confirmed that of the plaintiff's doctor.

Since in the medical mind the subarachnoiditis of the plaintiff could arise either from a negligent or non-negligent injection of the anesthetic, the burden cast upon the plaintiff to establish that the defendant was negligent simply is not met.

It would be pure speculation to say the defendant in injecting the anesthetic into the body of the plaintiff did so improperly. Judgments cannot rest on speculation or conjecture, there must be some substantial evidence for their support.

It is the contention of the plaintiff that the negligence of the defendant consisted of directly introducing into the subarachnoid space the caudal anesthetic which induced the arachnoiditis. It is admitted by the plaintiff that there is no direct evidence that the defendant did inject the anesthetic into the subarachnoid space. The plaintiff, however, contends that the circumstantial evidence is sufficient to establish a presumption of this fact. His argument may be summarized as follows:

(1) After the operation the plaintiff suffered from an arachnoiditis; (2) the arachnoiditis occurred because of the caudal anesthesia; (3) the defendant administered the anesthesia; (4) the direct introduction of a caudal anesthesia into the subarachnoid space is bad practice and can produce arachnoiditis; (5) therefore, a presumption may arise that the defendant was negligent and did inject this space.

The difficulty with this argument is that a physician is not a warrantor of a cure. His liability rests solely on accountability for his negligent or wrongful acts.

If the evidence in a case discloses two or more possible causes for an injury, for only one of which a defendant is responsible, liability does not attach unless the evidence discloses that the cause for which he is responsible is the more probable.

The judgment of the trial court was affirmed.

(Crewse v. Munroe, 11 CCH Neg. Cases 2d 1074-Oregon.)

Hospital Safety

Harriet L. Aberg, C.R.N.A.

"What are the real fire hazards in oxygen tent therapy?" is a perennial question. From time to time static studies on various textiles have been conducted, but few of these studies have been done under actual working conditions. Of course an oxygen tent fire, the same as any fire, depends on three basics, 1—something burnable, 2—oxygen, 3—heat (temperature high enough to flash the flammable material). Most of the studies have attempted to determine at just what point bedding will start to burn with static sparks and the sparks from electric appliances.

It is rather difficult to start a fire in a bed sheet with static sparks even in an oxygen enriched atmosphere. But change this just slightly to a realistic condition and the results differ. Hair, sheets, cotton balls and bandages having traces of oil on them can be ignited readily by a static spark in the atmosphere of an oxygen tent. There are many sources of this oil, such as skin treatments, compounds used for backrubs, and one which has been unrecognized till recently, hair tonic.

It isn't too difficult to eliminate oils in backrubs, skin treatments and such, but the hair tonic and skin and scalp oils are present when the patient is placed in an oxygen tent. Therefore, within the tent and its immediate surroundings, full protection must be taken and sources of ignition reduced.

The fan used in the mechanism of the tent should be electrically approved and properly adjusted so it can not throw sparks. It is best not to use wool blankets. Cotton uniforms for nursing personnel caring for patients in oxygen tents is recommended. The use of alcohol, oil, electric heating pads, call bell buttons, electric razors, flashlights, or similar equipment within the tent should be prohibited.

Miss Aberg is A.A.N.A.'s representative on the N.F.P.A. Committee on Hospital Operating Rooms.

Any questions pertaining to hospital safety may be directed to the Executive Office. Answers will be included in this section in future issues.

Abstracts

Clinical Anesthesia Conference, New York: Cardiovascular collapse in cardiac tamponade. New York State J. Med. 59: 3628-3630 (Oct. 1) 1959.

"Accumulation of fluid in the pericardium may become so excessive as to interfere with cardiac diastole. This condition can be aggravated by certain anesthetic maneuvers so as to result in cardiac tamponade. . . .

"A fifty-year-old nurse's aide was admitted to the hospital because of increasing shortness of breath and chest pain of three weeks duration. . . . It was felt that the patient had pericardial and pleural effusion of undetermined etiology. . . .

"Cardiac tamponade with disappearance of blood pressure occurred immediately after endotracheal intubation. Apnea developed following intravenous administration of succinylcholine and artificial control of respirations by intermittent manual compression of the breathing bag. Inquiry into the manner by which this controlled respiration was performed revealed that vigorous manual pressure, probably a pressure of 30 cm. of water, was being applied intermittently. Such vigorous bag pressure causes excessive pulmonary inflation.

"When this occurs in a patient with pericardial effusion while his chest is closed, the excess pulmonary inflation results in increased pressure on the pericardium, which may so constrict the heart as to interfere with cardiac filling. The result is what was observed in this case: arterial blood pressure disappears and the electrocardiogram displays a significant change, characterized by marked lowering both of the QRS complexes and the height of the T waves. Untreated, this would quickly result in death.

"The decision to carry on with the proposed pericardiostomy despite the critical condition of the patient was good. . . . Vigorous controlled respiration usually entails the application of at least 30 cm. of water pressure, and this should be avoided under such conditions."

Bellville, J. W., Howland, W. S. and Bross, I. D. J.: Postoperative nausea and vomiting. III. Evaluation of the antiemetic drugs fluphenazine (Prolixin) and promethazine (Phenergan) and comparison with trifluprom azine (Vesprin) and cyclizine (Marezine). J.A.M.A. 172: 1488-1493 (April 2) 1960.

"Postoperative nausea and vomiting is a frequent complication of anesthesia and surgery. Many compounds recommended for the treatment of this condition have been only casually studied, and little information on several compounds studied under similar conditions of control is available. To gain more insight into this problem as it exists at Memorial Center, [New York] we set up a controlled study in January, 1958. This report summarizes our results with various doses of the four compounds studied. . . . A total of 3,454 patients were studied. . . .

"Fluphenazine (Prolixin) and promethazine (Phenergan) are effective antiemetic agents. In comparison with

results obtained previously with administration of cyclizine (Marezine) and triflupromazine (Vesprin) they appear to be more effective than cyclizine and as effective as triflupromazine. In contrast to triflupromazine, clinically significant hypotensive side-effects were not seen after giving fluphenazine and promethazine in the dosage studied.

"A controlled, double-blind study was set up to study the effectiveness of these drugs in prophylactic treatment of postoperative nausea and vomiting. Selected patients, on arrival in the recovery room, received 1 ml. of a coded test drug intramuscularly and were carefully observed for 2½ hours by a full-time nurse observer. In the group of patients who received a placebo the incidence of nausea and vomiting was 18.5%. Cyclizine and triflupromazine which were studied initially reduced this incidence to about 11 and 5% respectively. In this study, giving fluphenazine and promethazine reduced the incidence to about 4%. Hypotensive side-effects were also evaluated. Significant prolongation of postanesthetic sleeping time was noted after giving promethazine but not after giving fluphenazine."

Dobell, A. R. C., Gutelius, J. R. and Murphy, D. R.: Acidosis following respiratory alkalosis in thoracic operations with and without heart-lung bypass. J. Thoracic and Cardiovascular Surg. 39: 312-317 (March) 1960.

"In the past decade anesthetists have become aware of the likelihood and the danger of carbon dioxide retention during anesthesia. The re-

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action to this knowledge has been a tendency to hyperventilate the patient. With respiration thus assisted by the anesthetist, depression of the arterial carbon dioxide tension may occur, particularly with the use of a muscle relaxant and complete control of ventilation by the anesthetist. This is particularly true in children. . . .

"The patients considered in this report fall into two groups—the 'pump-oxygenator' group and the 'control' group. The former consisted of 22 consecutive patients undergoing open heart surgery for a variety of congenital cardiac lesions using a screen-oxygenator. . . . The control group consisted of 8 children undergoing other thoracic operations. . . .

"When carbon dioxide reaccumulation was permitted at the end of operation, a sudden fall in pH occurred. The findings were similar in two groups of patients, one undergoing extracorporeal circulation for intracardiac surgery and the other having had other thoracic procedures. All but 2 of the patients were children."

Bullough, John: Use of premixed pethidine and antagonists in obstetrical analgesia, with special reference to cases in which levallorphan was used. Brit. M. J. 2: 859-862 (Oct. 31) 1959.

"The introduction into clinical practice of the two specific opiate antagonists nalorphine . . . and levallorphan . . . has opened up a new field in the relief of pain by morphine and all the drugs related to morphine. . . . The antagonists can be used to prevent respiratory depression occurring when an opiate is given, or to correct or relieve existing respiratory depression already caused by opiates. . . .

"A series of 130 patients consecutively admitted in early labour to one of two maternity wards were given intramuscular doses of pethidine 100 mg. premixed with levallorphan 2 mg., with the object of preventing neonatal respiratory depression. . . . Gas-air or 'trilene'-air was given in the later stages. Of the patients, 42% had two injections, 26% had one, one patient had nine, and the remainder (33%) had three to eight injections. Analgesia from the injections was good in 76%, poor or nil in 3%, and fair in the remainder. Of 48 patients questioned on the point, 60% preferred the injections to gas-air as more effective. . . .

"It is suggested that, by mixing an antagonist with pethidine, more of the latter drug can be given, providing better analgesia without causing an increased incidence of neonatal asphyxia. Relevant statistics from the maternity unit at Dartford over the years before and after the introduction of pethidine-antagonist mixtures are given, showed no evidence of adverse effects on mothers and babies."

Mond, Ernest and Mack, Irving: Cardiac toxicity of iproniazid (Marsilid): Report of myocardial injury in a patient receiving levarterenol. Am. Heart J. 59: 134-139 (Jan.) 1960.

"Various toxic effects of iproniazid have been recognized. These have been summarized in recent reports. Severe myocardial damage has, to our knowledge, not been observed. . . . A patient with iproniazid toxicity . . . developed the clinical and electrocardiographic picture of myocarditis after receiving intravenous levarterenol. . . . It is recommended that levarterenol and other pressor amines be used either not at all or with extreme caution in patients receiving iproniazid; they should never be used if toxicity to iproniazid is apparent."

Book Reviews

The Tonsils and Adenoids in Childhood. By Donald F. Proctor, M.D., Associate Professor of Laryngology and Otology; Assistant Professor of Physiology; Formerly Professor of Anesthesiology, The Johns Hopkins University School of Medicine. Charles C Thomas, Publisher, Springfield. Cloth. 70 pages, illustrations. Indexed. 1960. $7.50.

This book will be of particular interest to anesthetists since the author has not only a background of 25 years of association with the surgical aspects of tonsillectomy but also has had four years as Professor of Anesthesiology at the Johns Hopkins School of Medicine. Primarily devoted to the surgical aspects of tonsillectomy throughout the entire book, anesthetists will find useful information for the management of preoperative and postoperative care of the patient who is to have tonsillectomy and adenoidectomy. The subject of anesthesia is briefly covered as one phase in the surgical procedure. Liberal references follow each chapter in the book.

Anaesthetic Accidents. By V. Keating, M.B., B.Ch., D.A., F.F.A.R.C.S., Consultant Anaesthetist to the Manchester Regional Hospital Board (Burnley and District); formerly Consultant Anaesthetist and Lecturer in Anaesthetics, University College Hospital of the West Indies; Senior Specialist in Anaesthetics, Royal Army Medical Corps. The Year Book Publishers, Inc., Chicago. Cloth. 288 pages. Indexed. 2nd Ed., 1961. $5.50.

The second edition of this text has been extensively revised. Complications of both general and regional anesthesia are presented. The author has supplemented his personal experience with the reports of others. This book will be of value to all anesthetists who wish to be well informed.

Medical-Surgical Nursing. By Kathleen Newton Shafer, R.N., M.A., Formerly Associate Professor in Out-Patient Nursing, the Cornell University-New York Hospital School of Nursing, New York; Janet R. Sawyer, R.N., A.M., Instructor, School of Education, Department of Nurse Education, New York University, New York; Audrey M. McCluskey, R.N., M.A., Associate Professor in Nursing, the Cornell University-New York Hospital School of Nursing, New York; Edna Lifgren Beck, R.N., M.A., Associate Director of Nursing Education, Muhlenberg Hospital School of Nursing, Plainfield, N. J. The C. V. Mosby Company, St. Louis. Cloth. 876 pages, 141 illustrations, indexed. 2nd Ed., 1961. $8.75.

In this edition new material has been included. However, emphasis remains on nursing care of the patient. The book is divided into two sections. The first deals with general considerations. The second considers nursing related to specific medical and surgical care.

Of special interest to anesthetists are the chapters on fluid and electrolyte balance, preoperative and postoperative care, and the patient receiving anesthetics.

Many excellent illustrations are included. An extensive list of references follows each chapter.

Classified Advertisements

NURSE ANESTHETIST, Male or Female, for hospital on Staten Island, N. Y., excellent conditions. Write: Box B-44, Journal American Association of Nurse Anesthetists, Prudential Plaza, Suite 3010, Chicago 1, Ill.

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WANTED: Female or Male Anesthetist, CRNA member with experience. Opening on March 15, 1961 in 80 bed hospital, all modern equipment, located 30 miles from Houston, Texas. Salary —$600.00 per month, Surgery Anesthesia only, staff of twelve Doctors. Four room apartment available on hospital grounds; rent reasonable. Contact Wm. S. Nichols, Administrator, Polly Ryon Memorial Hospital, Richmond, Texas.

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ANESTHETIST: Position open in 360 bed hospital in Jacksonville, Florida, located on the St. Johns River. 40 hour week, rotating Surgery and OB. Liberal Personnel Policy. Please send replies to the Director of Personnel, St. Vincent's Hospital, Barrs and St. Johns Ave., Jacksonville 4, Fla.

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REGISTERED NURSE ANESTHETIST—Male or Female, fully accredited modern 150 bed hospital, department directed by Chief of Surgery. Starting salary $7,200.00 per year, plus two weeks vacation, health insurance available at nominal cost, sick leave. Social Security and group life insurance, paid educational leave, also full maintenance will be furnished, in addition to salary, should Anesthetist desire to live in our dormitory. Apply to: Homer E. Allen, Administrator, Clinch Valley Clinic Hospital, Richlands, Va.

NURSE ANESTHETIST. Holy Cross Hospital, 4777 E. Outer Drive. Detroit 34, Mich.

MARRIED MALE C.R.N.A. desires position with a fee for service basis and a guaranteed minimum salary. Am interested in the West or Southwest portion of the United States. Reply: Box B-64, Journal American Association of Nurse Anesthetists, Prudential Plaza, Suite 3010, Chicago 1, Ill.

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NURSE ANESTHETIST — Accredited 380 bed General Hospital. Modern air-conditioned facilities. Department consists of a Head Nurse Anesthetist and 6 staff members. Call is evenly rotated. Salary open depending upon experience. Liberal employee benefits. Apply: Miss Lucy Richards, Head Nurse Anesthetist, Lutheran Hospital, 2609 Franklin Blvd., Cleveland 13, Ohio.

Hicks

(Continued from page 79)

pharmacology involved and by the penetrating history by all concerned of the medications that the preoperative elective surgical patient is taking prior to the scheduling of such patients for operation. It is recommended that such cases be withdrawn from rauwolfia therapy 2 weeks prior to scheduling of elective surgery. Surgical emergencies must obviously be handled and it is for these cases that we should reserve norepinephrine in controlling the hypotensive status.

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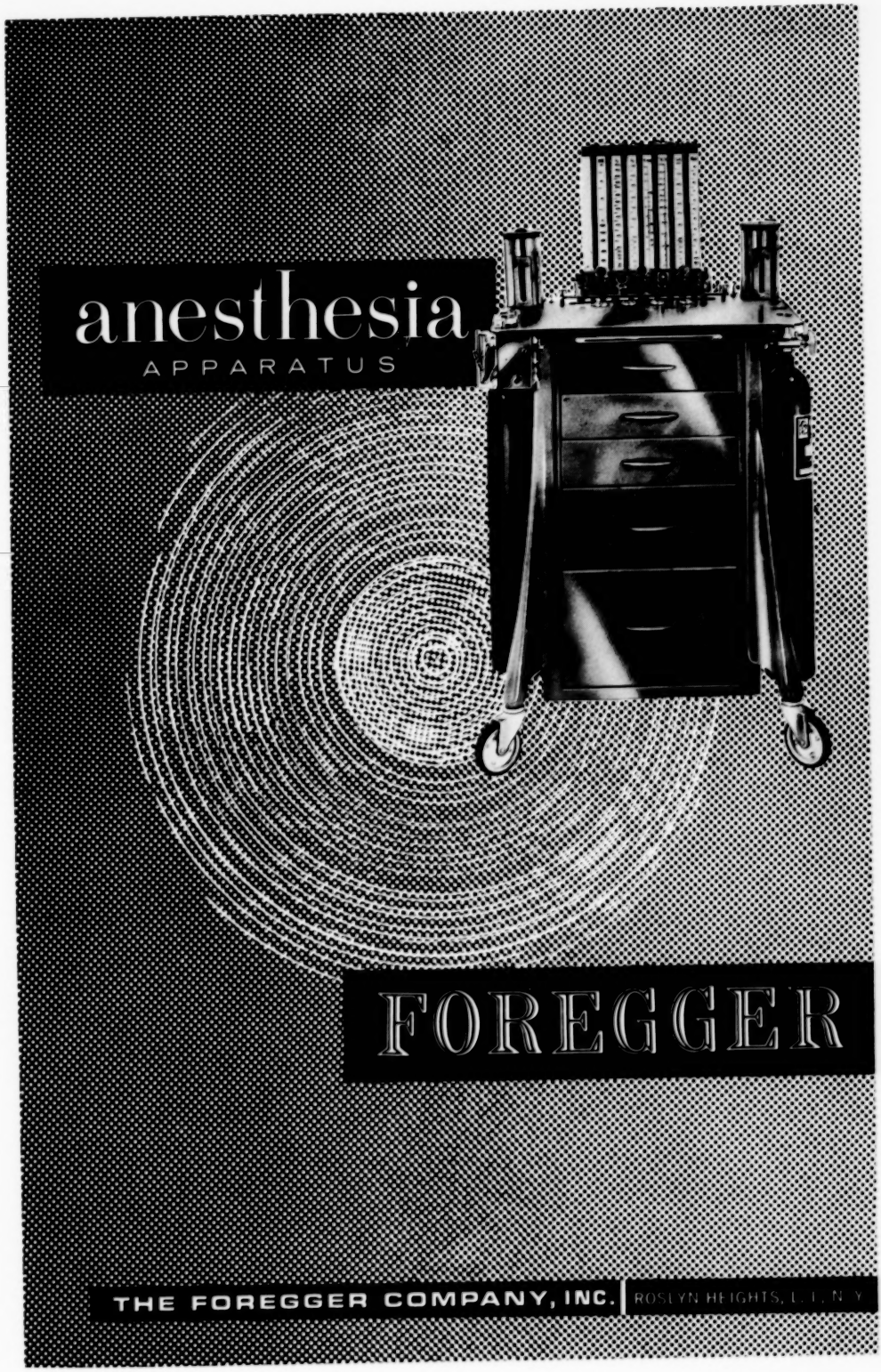
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